important factor in the pathogenesis of the articular disease.

Summary

In a young woman with psoriasis and no previous arthritis, varicella was associated with arthritis affecting the DIP joints in the pattern of classic psoriatic arthritis. The rapid clearing of the arthritis with resolution of the varicella suggests that the virus may have triggered the arthritis in susceptible joints.

REFERENCES

1. Bennett JC: The infectious etiology of rheumatoid arthritis. Arthritis Rheum 21:531-538, Jun 1978
2. Arthritis caused by viruses—Medical Staff Conference, University of California, San Francisco. Calif Med 119:38-44, Sep 1973
3. Mulhern LM, Friday GA, Perri JA: Arthritis complicating varicella infection. Pediatrics 48:827-829, Nov 1971
4. Ward JR, Bishop B: Varicella arthritis. JAMA 212:1954-1956, Jun 1970
5. Leonard DG, O'Duffy JD, Rogers RS: Prospective analysis of psoriatic arthritis in patients hospitalized for psoriasis. Mayo Clin Proc 53:511-518, Aug 1978
6. Moll JMH, Wright V: Psoriatic arthritis. Semin Arthritis Rheum 3:55-78, 1973

Refer to: Warren SE, Boice JB, Bloor C, et al: The athletic heart revisited—Sudden death of a 28-year-old athlete. West J Med 131:441-447, Nov 1979

The Athletic Heart Revisited

Sudden Death of a 28-Year-Old Athlete

LCDR SANFORD E. WARREN, MC, USNR
LT JAMES B. BOICE, MC, USNR
COLIN BLOOR, MD
CAPT W. V. R. VIEWEG, MC, USN
San Diego

IN THE PAST *organic heart disease* was often the diagnosis when an asymptomatic, trained athlete presented with electrocardiographic evidence of left ventricular hypertrophy and a large heart shown on x-ray studies. In recent years, however, the diagnosis is more likely to be *athletic heart syndrome*.[1-5] This increasingly popular diagnosis is usually applied to young athletes capable of championship performance who are found to have evidence of cardiac hypertrophy and enlargement, a slow resting heart rate, systolic ejection murmur and, occasionally, a variety of arrhythmias and conduction disturbances.[1] This condition has been thought adaptive, benign and reversible, and restrictions on physical activity were not thought to be indicated.[6,7] Nevertheless, occasional reports of sudden death in marathon runners with patent coronary arteries[8,9] and the knowledge that only a history of athletic prowess deters a diagnosis of cardiomyopathy in a patient with unexplained cardiac enlargement, calls into question the presumed benign nature of this condition. Moreover, the adaptive response of a normal heart to isotonic and isometric exercise differs, and it is not known under what circumstances (if any) the response may become pathologic.

Just as a patient with the athletic heart syndrome is asymptomatic at diagnosis, so too may be persons from a kindred destined to manifest familial cardiomyopathy, who often have a subsequent malignant course characterized by congestive heart failure or sudden death.[10-12] If these patients were initially capable of competitive physical exercise, it is possible that a spurious diagnosis of athletic heart syndrome would be made and the person encouraged to pursue limitless physical exertion, perhaps at some hazard.

In this paper we report the case of a young national champion tennis player who carried the diagnosis of athletic heart syndrome for six years following a complete cardiac evaluation in 1972. In this woman and several members of her family, all athletically active, concentric left ventricular hypertrophy was shown by echocardiography. The woman collapsed while playing tennis and died within 24 hours. On autopsy there was evidence of acute myocardial infarction, patent coronary arteries and diffuse cardiomyopathy.

Reports of Cases

CASE 1. At the time of initial evaluation in 1972, the then 23-year-old patient had a negative past medical history and had participated competitively in a variety of sports including tennis, field hockey, basketball, volleyball, swimming and track. Her best time in the 100 yard dash was 11.9 seconds, in the 440 yard dash 61.1 seconds and in the two mile run 12 minutes 36 seconds. She was a recent singles runner-up in the All-Navy Tennis Tournament. The patient

From the Cardiology Division, Department of Internal Medicine and the Department of Pathology, Naval Regional Medical Center, San Diego, and the Department of Pathology, University of California, San Diego, School of Medicine, San Diego.

Supported in part by Bureau of Medicine and Surgery Clinical Investigation Program, Department of the Navy.

Submitted, revised, February 7, 1979.

The opinions or assertions contained herein are those of the authors and are not to be construed as official or reflecting the views of the Department of the Navy.

Reprint requests to: Capt. W. V. R. Vieweg, MC, USN, Head, Cardiology Branch, Naval Regional Medical Center, San Diego, CA 92134.

desired to be a naval aviator and was referred to the Cardiology Department, Naval Regional Medical Center, San Diego, California, after a routine x-ray study of the chest and an electrocardiogram showed left ventricular prominence and hypertrophy (Figures 1 and 2A).

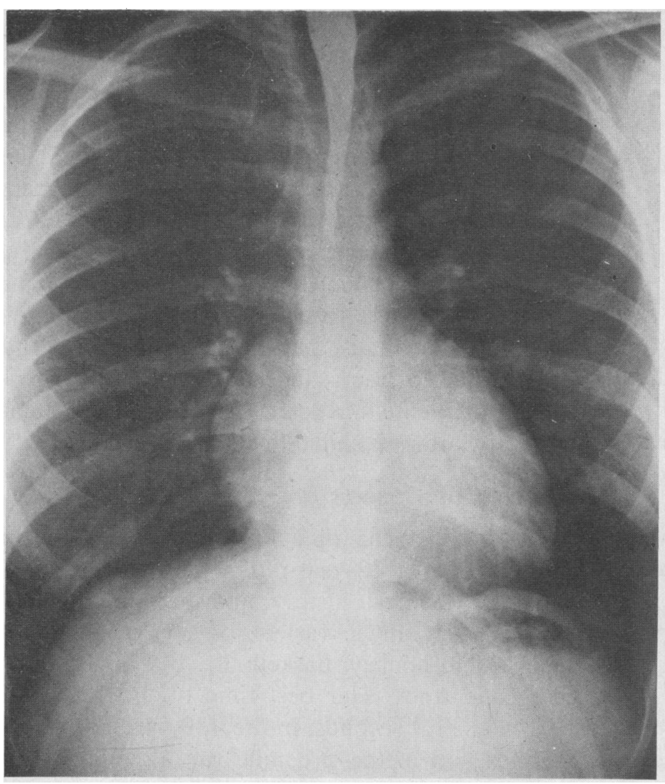
Physical examination disclosed a resting pulse of 50, blood pressure 120/75/55 mm of mercury, a grade I/VI systolic ejection murmur at the left sternal border, and a third heart sound. A treadmill test done according to the protocol by Bruce[13] was stopped after 18 minutes because the patient became fatigued, a maximum heart rate of 200 beats per minute having been achieved. No chest pain occurred; the postexercise tracing is shown in Figure 2B. An echocardiogram (Figure 3 and Table 1) disclosed increased thickness of the ventricular septum and posterior wall of the left ventricle with a normal ratio and normal left ventricular function. Findings at cardiac catheterization included a symmetrically thickened ventricle, mildly elevated right and left ventricular end-diastolic pressures (11 and 17 mm of mercury), an ejection fraction of 83 percent, normal coronary arteries, and no valvular disease or gradients. Hypertrophic subaortic stenosis (IHSS) was excluded by postpremature ventricular contraction pressure changes and isoproterenol infusion. A diagnosis of athletic heart syndrome was made.

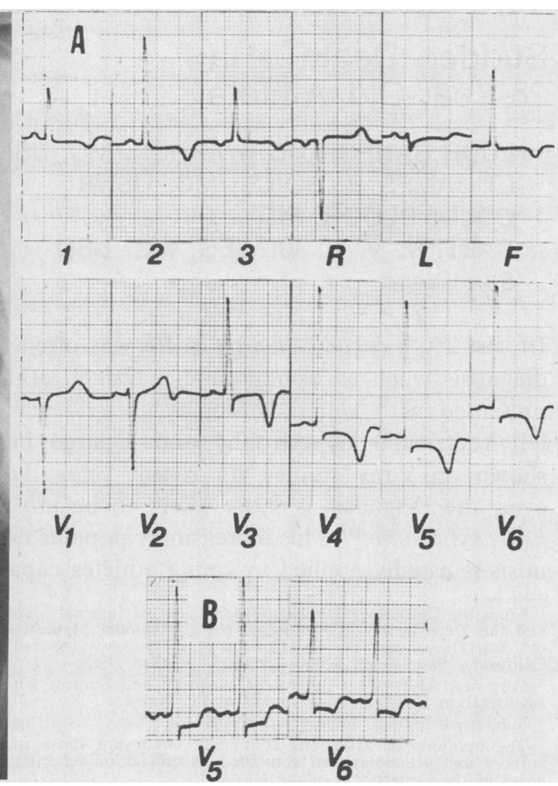
Several months later the patient embarked on a professional tennis career, playing competitively until 1976. At that time, she experienced the onset of frequent 15- to 30-minute episodes of crushing substernal chest pains associated with shortness of breath, nausea and diaphoresis, occurring at rest. The frequency of discomfort remained stable at every other day, and the patient continued to play tennis and jog 1 to 3 miles a day.

In February 1978 (at age 28), the patient collapsed shortly after beginning a tennis match; she was resuscitated, but died within 24 hours. Her brief time in hospital was complicated by clinical, electrocardiographic and enzymatic evidence of acute anteroseptal myocardial infarction, adult respiratory distress syndrome and malignant ventricular arrhythmias.

Autopsy Findings

At necropsy, the heart weighed 500 grams and showed left ventricular prominence. The atria were mildly dilated. Transverse sections of the heart showed bilateral, diffuse, concentric hypertrophy





Figures 1 and 2.—Roentgenogram of the chest and electrocardiogram in an outstanding 23-year-old female athlete showing left ventricular hypertrophy. Item B in Figure 2 is the postexercise electrocardiogram.

which was more pronounced in the left ventricle. Although the right ventricle had a normal chamber size, the pronounced left ventricular hypertrophy significantly reduced left ventricular chamber size. The anterior, posterior and lateral walls of the left ventricle and interventricular septum were equally involved, averaging about 2.8 cm in thickness (Figure 4). The interventricular septum/posterior wall thickness ratio of the left ventricle was 1.0. At its point of maximum thickness the right ventricular free wall measured 0.9 cm. The coronary arteries and valves were normal.

At the left ventricular apex extensive regions of circumferential subendocardial infarct were noted. More cephalad, there were multifocal infarcts involving all regions of the ventricular wall, predominantly in the inner half. Several regions of old infarct were present in the posterior apical wall of the left ventricle.

Histologically, the zones of gross infarct showed characteristic changes including fiber disruption and cellular infiltrate consistent with an infarct of 24 to 48 hours duration. Sections of the coronary arteries were normal (Figures 5 and 6). Other

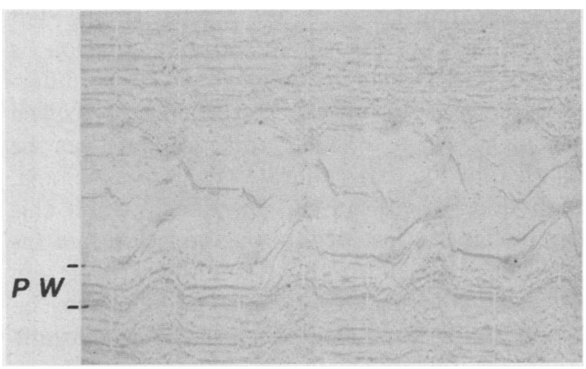


Figure 3.—Echocardiogram in a 23-year-old female athlete showing concentric hypertrophy with posterior wall (PW) thickness of 14 mm in diastole. Septal measurements were determined from other portions of the echocardiogam.

myocardial lesions comprised diffuse interstitial broad band fibrosis and myocardial fiber hypertrophy which were present in sections from the walls of both ventricles. No changes of amyloidosis, sarcoidosis, storage disease or rheumatic heart disease were present.

Case histories of family members are given below.

CASE 2. The father of the patient is a 60-year-old former semiprofessional athlete who was active in track, basketball, swimming and tennis. An excellent tennis player all his life, in recent years he has confined tennis to doubles because of eye surgical procedures. He has had no symptoms of cardiovascular disease, but gives a history of having "a large heart" all his life. During evaluation in 1972, he was found to have a resting pulse of 50, blood pressure 175/90/90 mm of mercury and a grade II/VI systolic ejection murmur at the left sternal border. An x-ray study of the chest showed left ventricular prominence and an electrocardiogram indicated left ventricular hypertrophy (Figures 7 and 8A). He exercised seven minutes on the treadmill[13] and achieved a maximum heart rate of 135 beats per minute before the test was stopped due to shortness of breath. The postexercise tracing appears in Figure 8B. Echocardiographic results appear in Table 1.

CASE 3. The 33-year-old brother of the patient in the index case is an aviator with an outstanding athletic record. In 1972 an x-ray study of the chest and an electrocardiogram showed left ventricular prominence. During evaluation in 1972 he completed 18 minutes on the treadmill without chest pain, arrhythmia, or significant postexercise ST segment changes developing. Echocardiographic results appear in Table 1.

CASE 4. The 31-year-old married sister of

TABLE 1.—*Echocardiographic Profile of an Athletic Family*

	EDD	*ESD*	*SV*	*EF*	*Vcf*	*Δd/d*	*S&PWT*
Normal	35-53			59-75	1-1.5	32-39	8-11
Case 1 (1972) Index case	47	28	82	79	1.26	40	14
Case 2 (1978) Father	42	24	60	81	1.20	43	14
Case 3 (1978) Brother	52	32	108	77	1.75	58	13
Case 4 (1978) Sister	43	26	62	78	1.13	40	9
Case 5 (1978) Mother	41	24	55	80	1.12	41	9

EDD = end-diastolic dimension (in millimeters)
ESD = end-systolic dimension (in millimeters)
SV = stroke volume (in cubic centimeters)
EF = ejection fraction (in percent) (determined by cubing the short diameter)
Vcf = mean velocity of circumferential fiber shortening (in circumferences per second)
Δd/d = fractional shortening (in percent)
S&PWT = septal and posterior wall thickness at end diastole (in millimeters)

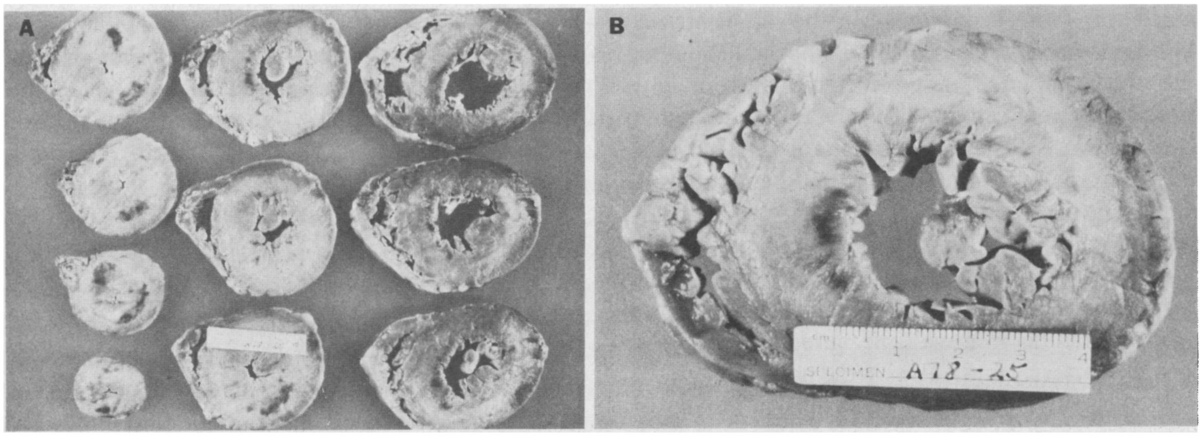


Figure 4.—Concentric hypertrophy, myocardial fibrosis and acute and old myocardial infarctions in a 28-year-old female champion athlete. Darkened areas show fresh subendocardial infarction.

Figure 5.—Microscopic section of anterior left ventricular myocardium using hematoxylin-eosin stain. Entire field shows infarction with an interstitial leukocytic infiltrate.

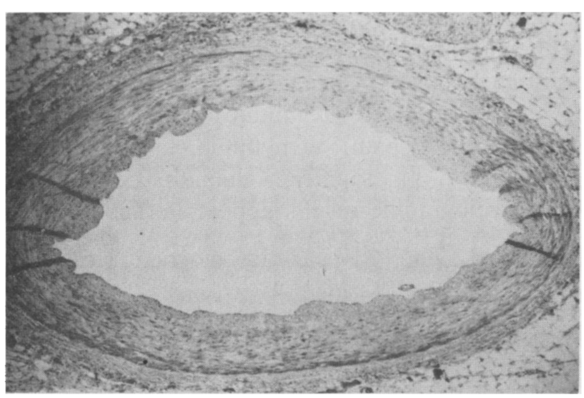


Figure 6.—Normal left anterior descending coronary artery representative of remaining coronary system.

the patient was an outstanding athlete in high school, but subsequently injured her knee and retired from competitive sports. An x-ray study of the chest, electrocardiogram and echocardiogram are normal. Echocardiographic results appear in Table 1.

CASE 5. The 62-year-old mother has no history of competitive athletics or heart disease. Physical examination, electrocardiogram and x-ray study of the chest showed no abnormalities. Echocardiographic results appear in Table 1.

Discussion

The myocardial response to exercise has been studied extensively.[14-19] While the resting left ventricular function of a sedentary person is comparable to that of a trained athlete,[19] differences emerge during exercise. Increased oxygen needs, hence increased cardiac output, can be supplied by increases in heart rate, stroke volume, or both of these. In an untrained person this requirement is met chiefly by the heart rate increasing, while in an athlete both heart rate and stroke volume are utilized.[15,16] The latter may be due partially to increased cardiac end-diastolic volume.[17] Moreover, in an athlete there is an enhanced ability to extract oxygen peripherally.[18]

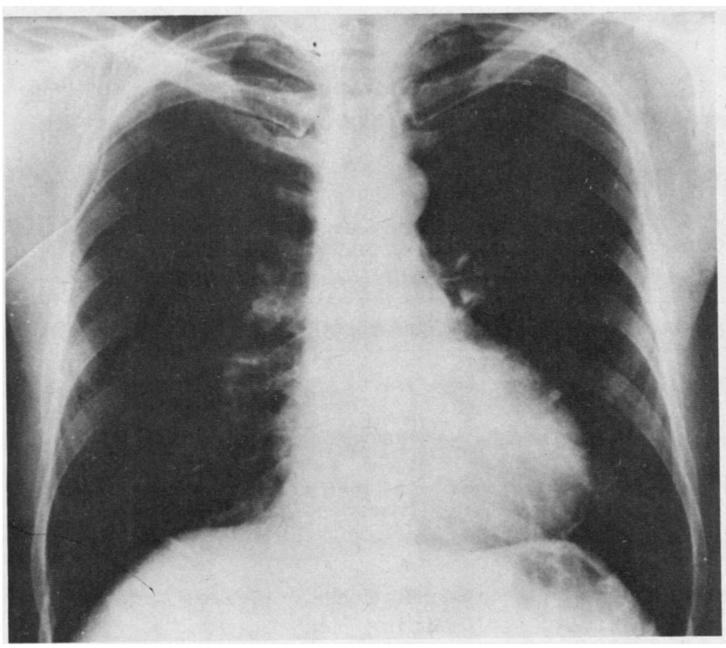
Both chronic isometric exercise and isotonic exercise result in increased left ventricular mass, while isotonics also produce increased ventricular volume.[3,14,22] In an athlete these changes are brought about by an enlargement of myocardial fiber size rather than number, and occur without accompanying fibrosis (as in the volume stress of regurgitant valvular or primary myocardial disease; see Table 2).[20-22] There has been doubt as to the universal adequacy of capillary flow to match an enlarging myocardial mass,[23] such that stressed muscle fibers might ultimately suffer nutritional deficiency and hypoxia,[24] or perhaps be prone to failure. In fact, elevated end-diastolic pressures have been recorded in young athletes,[7] as well as sudden death with patent coronary vessels.[8,9]

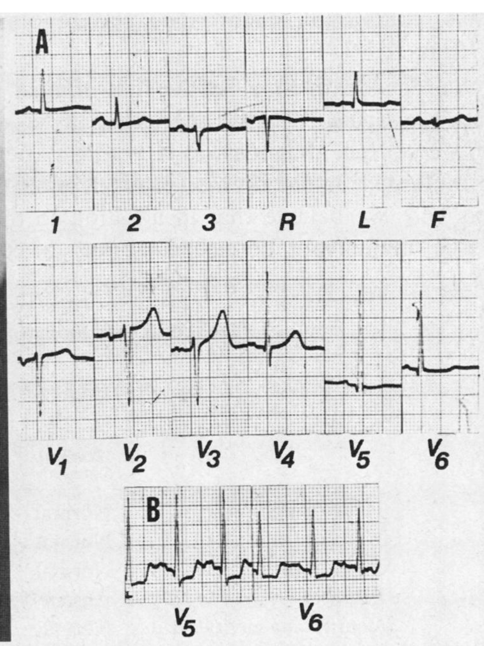
Fortunately, from a clinical standpoint, the heart has never been shown to outgrow its own blood supply, with the possible exception of cases of congenital single coronary artery.[25] Could familial cardiomyopathy be a second example, at least in the presence of exercise? This may have been the case in our patient.

The patient in the index case reported in this paper presented without symptoms in 1972, and evidence of left ventricular hypertrophy was noted. Secondary causes of ventricular hypertrophy such as hypertensive cardiovascular disease, aortic stenosis and idiopathic hypertrophic subaortic stenosis were excluded at catheterization and autopsy; nor were secondary causes of diffuse myocardial disease found to be present. A diagnosis of familial cardiomyopathy was entertained[10-12] in light of the presence of several similarly affected family members, but this diagnosis was not established because of several considerations.

First, a review of 11 families with 47 members showing familial cardiomyopathy disclosed a mean age at diagnosis of 24 years, with most cases showing symptoms by the fifth decade.[10] Our patient's asymptomatic father in his 60's militated against such a process. Second, while familial cardiomyopathy may be asymptomatic at discovery,[10] enhanced performance is not seen.[11,12] To our knowledge, in fact, there are no well-documented reports in the literature showing championship performance in a patient destined to die because of familial cardiomyopathy. Third, the criteria of Gott and coworkers[1] for the diagnosis of the athletic heart syndrome were fulfilled: namely, a history of athletic endurance, biventricular enlargement, systolic ejection murmur, third heart sound, resting sinus bradycardia and increased indices of cardiac function.

In retrospect, we believe that the patient in the index case probably had a variant of familial cardiomyopathy[26] with some features of the athletic heart syndrome. Her principal forms of exercise were isotonic. Yet, her echocardiogram in 1972 showed left ventricular hypertrophy without dilatation as did her father's and brother's echocardiograms in 1978. Often there is echocardiographic evidence of both left ventricular hypertrophy and dilatation in an endurance-trained athlete.[18,29] Her resting electrocardiogram showed repolarization changes consistent with left ventricular strain (pressure or systolic overload). In contrast, the usual resting electrocardiogram in a trained athlete shows early repolarization with or without tall T-waves (volume or diastolic overload).[27] Following treadmill exercise testing there was ST segment flattening and depression of greater than 2 mm consistent with an *"ischemic*





Figures 7 and 8.—Roentgenogram of the chest and electrocardiogram showing left ventricular hypertrophy in a 60-year-old former champion athlete (case 2). Item B in Figure 8 is the postexercise electrocardiogam.

response" in both the patient (case 1) and her father (case 2). Such postexercise electrocardiographic changes are rarely seen in the athletic heart syndrome.[22]

Postmortem examination in the index case showed striking evidence of left ventricular hypertrophy which presumably progressed over the preceding years. The echocardiogram in 1972 showed septal and posterior wall thickness of 14 mm during diastole in comparison to 28 mm at necropsy in 1978. Even if the heart had arrested at end-systole, there had been significant progression of hypertrophy. In addition to hypertrophy there was evidence of old and new myocardial infarcts in the presence of patent coronary arteries. Clearly, either myocardial perfusion was inadequate to meet the demands of the hypertrophied heart or myocardial flow was redistributed, making the subendocardial zones more vulnerable to ischemic damage.

Because most reviews and case reports of the athletic heart syndrome fail to provide long follow-up or to examine other family members, it is possible that some athletes so labelled do, in fact, have underlying heart disease that will become manifest at a future date. For any individual case it may be difficult to differentiate cardiomyopathy from the athletic heart syndrome. In addition to cardiac enlargement and hypertrophy, ventricular gallop and systolic ejection murmur, both conditions may show a variety of arrhythmias, conduction disturbance and repolarization changes.[6,10,27]

Sudden death may be the first clinical sign of cardiomyopathy[28] and, therefore, it is obvious that a correct diagnosis in a young patient with an enlarged, hypertrophied heart is essential. To this end, we believe that in addition to the diagnostic maneuvers described above, a complete history with examination of family members should be carried out. If cardiomegaly on x-ray films of the chest, left ventricular hypertrophy on electrocardiograms and measurably increased wall thickness on echocardiograms are found in family members (especially if not athletic), great caution should be exercised before a diagnosis of athletic heart syndrome is applied. Even if, as in our case, affected family members have been athletic, due to the highly variable clinical expression of familial cardiomyopathy[10] the patient and relatives should receive close follow-up.

Summary

A young female athlete capable of championship performance presented in 1972 with left ventricular prominence, left ventricular hypertrophy with strain and concentric left ventricular hypertrophy on x-ray studies of the chest, electrocardiogram and echocardiogram, respectively. Several family members, including her asymptomatic 60-year-old father, were also found to possess both athletic histories and large hearts. In view of the history and asymptomatic status of older family members, a diagnosis of athletic heart syndrome was made. Six years later the patient suffered a cardiac arrest during exercise, and an autopsy showed evidence compatible with familial cardiomyopathy. It is concluded that patients with familial cardiomyopathy may be capable of competitive physical endurance initially and that physicians should defer a diagnosis of athletic heart syndrome in athletic patients with large hearts until proper diagnostic tests and family studies are done.

REFERENCES

1. Gott PR, Rosselle HA, Crampton RS: The athletic heart syndrome—Five-year cardiac evaluation of a champion athlete. Arch Intern Med 122:340-344, 1968

2. Grande F, Taylor HL: Adaptive changes in the heart, ves-

TABLE 2.—*Pathologic Spectrum of Myocardial Hypertrophy*[20-22]

	Normal Sedentary	*Normal Athlete*	*Pathologic Concentric (Early Pressure Loads)*	*Pathologic Eccentric (Valvular Regurgitation, Myocardial Disease Excluding IHSS)*
Heart weight	Normal	↑ but<500 grams	≥500 grams	≥500 grams
Number of fibers	Normal	Normal	Normal	↓
Fiber size	Normal	↑	↑	↑↑
Fibrosis	Absent	Absent	Generally absent	Present
Ventricular cavity size	Normal	Normal or ↑; ↓ in systole	Normal or ↓	↑

↑ increased ↓ decreased ↑↑ greatly increased IHSS = hypertrophic subaortic stenosis

sels, and patterns of control under chronically high loads, *In* Hamilton WF, Dow P (Eds): Handbook of Physiology: Circulation, Vol III. Washington, DC, 1965, Am Physiol Soc, pp 2615-2677

3. Roeske WR, O'Rourke RA, Klein A, et al: Noninvasive evaluation of ventricular hypertrophy in professional athletes. Circulation 53:286-292, 1976

4. Morehouse LE, Miller AT Jr: Physiology of Exercise, 5th Ed. St. Louis, The C V Mosby Co, 1967, pp 93, 286, 287

5. Pyorala J, Kyronseppa H: Cardiovascular studies on former endurance athletes, *In* Karvonen MJ, Barry AJ (Eds): Physical Activity and the Heart. Springfield, IL, Charles C Thomas, 1967, pp 301-310

6. Lichtman J, O'Rourke RA, Klein A, et al: Electrocardiogram of the athlete. Arch Intern Med 132:763-770, 1973

7. Marcano BA, Moss AJ: Spurious heart disease in athletic children. J Pediatr 72:664-668, 1968

8. Green LH, Cohen SI, Kurland G: Fatal myocardial infarction in marathon racing. Ann Intern Med 84:704, 1976

9. Lollgen H, Just HJ, Mathes P: Myokardinfarkt bei einem Hochleistungs-Sportler mit normalen Koronarterien. Dtsch Med Woch 98:620, 1973

10. Kariv I, Kreisler B, Sherf L, et al: Familial cardiomyopathy—A review of 11 families. Am J Cardiol 28:693-706, 1971

11. Evans W: Familial cardiomyopathy. Br Ht J 2:68-82, 1949

12. Boyd DL, Mishkin ME, Feigenbaum H, et al: Three families with familial cardiomyopathy. Ann Intern Med 63:386-401, 1965

13. Doan AE, Peterson DR, Blackman JR, et al: Myocardial ischemia after maximal exercise in healthy man. Am Heart J 69: 11:21 1965

14. Wenger NK, Gilbert CA: Athletes heart, *In* Hurst JW (Ed): The Heart. New York, McGraw-Hill Book Co, 1978, pp 1706-1709

15. Rushmer RF, Smith O: Cardiac control. Physiol Rev 39: 41, 1959

16. Rushmer RF, Smith O, Franklin D: Mechanism of cardiac control in exercise. Circ Res 7:602 1959

17. Sarnoff ST, Mitchell JH: The control of the function of the heart, *In* Hamilton WF (Ed): Handbook of Physiology: Circulation, Vol I. Washington, DC, American Physiological Society, 1962, p 525

18. Bevegard S, Holmgren A, Jonsson B: Circulatory studies in well-trained athletes at rest and during heavy exercise, with special reference to stroke volume and the influence of body position. Acta Physiol Scand 57:26-50, 1963

19. Gilbert CA, Nutter DO, Felner JM, et al: Echocardiographic study of cardiac dimensions and function in the endurance-trained athlete. Am J Cardiol 40:528-533, 1977

20. Badeer HS: Biological significance of cardiac hypertrophy (Editorial). Am J Cardiol 14:133-138, 1964

21. Allen WD, Goldberg SJ, Sahn DJ, et al: A quantitative echocardiographic study of champion childhood swimmers. Circulation 55:142-145, 1977

22. Raskoff WJ, Goldman S, Cohn K: The athletic heart. JAMA 236:158-162, 1976

23. Honig CR, Bourdeau-Martin J: Extravascular component of oxygen transport in hypertrophied hearts with special reference to oxygen therapy. Circ Res 35 (Suppl II):II-97, 1974

24. Linzbach AJ: Heart failure from the point of view of quantitative anatomy. Am J Cardiol 5:370, 1960

25. Warren SE, Alpert JS, Vieweg WVR, et al: Normal single coronary artery and myocardial infarction. Chest 72:540-543, 1977

26. Perloff JK: The cardiomyopathies—Current perspectives. Circulation 44:942-949, 1971

27. Hanne-Paparo N, Drory Y, Schoenfeld Y, et al: Common ECG changes in athletes. Cardiology 61:267-278, 1976

28. Maron BJ, Roberts WC, Edwards JE, et al: Sudden death in patients with hypertrophic cardiomyopathy: Characterization of 26 patients without functional limitation. Am J Cardiol 41:803-810, 1978

29. DeMaria AN, Neumann BS, Garrett L, et al: Alterations in ventricular mass and performance induced by exercise training in man evaluated by echocardiography. Circulation 57:237-243, 1978

Clinical Depression and Suicide

THERE EXISTS a powerful clinical bias not to understand depression as an illness, but rather to explain it away. A recent study of completed suicides makes two striking and uncomfortable observations: . . . the vast majority of these patients were under the recent care of a physician, and . . . a review of their records showed that clinicians had documented sufficient signs and symptoms to make a diagnosis of clinical depression . . . without having done so.

—HENRY R. BLEIER, MD, *Philadelphia*

Extracted from *Audio-Digest Internal Medicine,* Vol. 26, No. 12 in the Audio-Digest Foundation's subscription programs. For subscription information: 1577 E. Chevy Chase Drive, Glendale, CA 91206